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Asymptomatic traumatic rupture of an intracranial dermoid cyst: A case report

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Abstract

BACKGROUND

Previous studies reported that most of the intracranial dermoid cyst ruptures were spontaneous, and only a few were traumatic, with asymptomatic much rarer than the symptomatic ruptures. Hence, how to deal with the asymptomatic traumatic rupture of intracranial dermoid cyst remains a challenge in the clinic.

CASE SUMMARY

A 59-year-old man was accidentally diagnosed with intracranial dermoid cyst through a cranial computed tomography (CT) scan due to a car accident. A mixed-density lesion with fat and a calcified margin was observed in the midline of the posterior fossa, accompanied with lipid droplet drifts in brain sulci, fissures, cisterns, and ventricles. After 1 wk of conservative observation, no change was observed on the updated cranial CT scan. After 2 wk of conservative observation, magnetic resonance imaging examination confirmed that the lesion was a traumatic rupture of a posterior fossa dermoid cyst with lipid droplet drifts. As the patient exhibited no adverse symptoms throughout the 2 wk, a 6-mo follow-up visit was arranged for him instead of aggressive treatment. Nonetheless, the patient did not show any abnormal neurological symptoms in the 6 mo of follow-up visits.

CONCLUSION

Asymptomatic traumatic rupture of intracranial dermoid cyst could be just followed or treated conservatively rather than treated aggressively.

Key Words: Intracranial dermoid cyst; Magnetic resonance imaging; Computed tomography; Rupture; Traumatic; Case report

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Core Tip: A case of traumatic intracranial dermoid cyst rupture in a 59-year-old man was analyzed retrospectively. Imaging findings, clinical manifestations, treatment, and follow-up results for 6 mo were collected. Combined with the relevant literature, the cause and treatment of asymptomatic cyst rupture were analyzed. For a traumatic intracranial dermoid cyst rupture, if the lesion is small, affects the adjacent structures only slightly, and has few lipid droplet drifts, conservative treatment or only follow-up visits could be adopted according to the patient's neurological examination results.

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INTRODUCTION

Intracranial dermoid cyst is a rare embryonal benign cystic mass and accounts for 0.04%-0.6% of intracranial tumors[1,2]. Previous studies reported that most of the intracranial dermoid cyst ruptures were spontaneous[2-5], and only a few were traumatic[1,6], with asymptomatic much rarer than the symptomatic ruptures. The current study aimed to comprehensively analyze the imaging characteristics of traumatic dermoid cyst rupture based on the existing case and literature, thereafter the intervention was determined based on the imaging characteristics and clinical symptoms.

CASE PRESENTATION

Chief complaints

A 59-year-old man was admitted to the hospital due to a car accident.

History of present illness

A cranial computed tomography (CT) scan revealed a mixed-density lesion near the midline of the posterior fossa (anterior and superior to the occipital protuberance), accompanied by a calcified margin.

Physical examination

No other traumatic lesions, such as intracranial hemorrhage and skull fracture, were observed (Figure 1). As the patient had no obvious neurological symptoms, he was discharged and sent home for recuperation.

Imaging examinations

The CT value in the low-density lesion area was about 14 Hounsfield Units (HU) and that in the fat-density area was -60 to -40 HU; the size of the lesion was 14.6 mm × 21.1 mm without a space-occupying effect. In addition, low-density spotted shadows were observed in the ambient cistern, interpeduncular cistern, longitudinal fissure cistern, juxta-right cavernous sinus, lateral ventricles, and sulus; the CT value was about -60 to -40 HU, and the maximum length of the lipid droplets was 5 mm.

On the updated cranial CT scan after 1 wk, no changes were detected in the lesion or the distribution or the size of the lipid droplets as compared to that at the first admission. After 2 wk of conservative observation, the patient underwent cranial magnetic resonance imaging (MRI) examination, in which the lesion was located below the cerebellar vermis, with the anterior part extending into the fourth ventricle. As a result, hyperintensity was detected on T2 weighted image, hypointensity on T1-fluid attenuated inversion recovery (FLAIR), iso-hypointensity on T2-FLAIR, and hyperintensity on diffusion weighted image ($b = 1000 \text{ s/mm}^2$). A T1-FLAIR contrast-

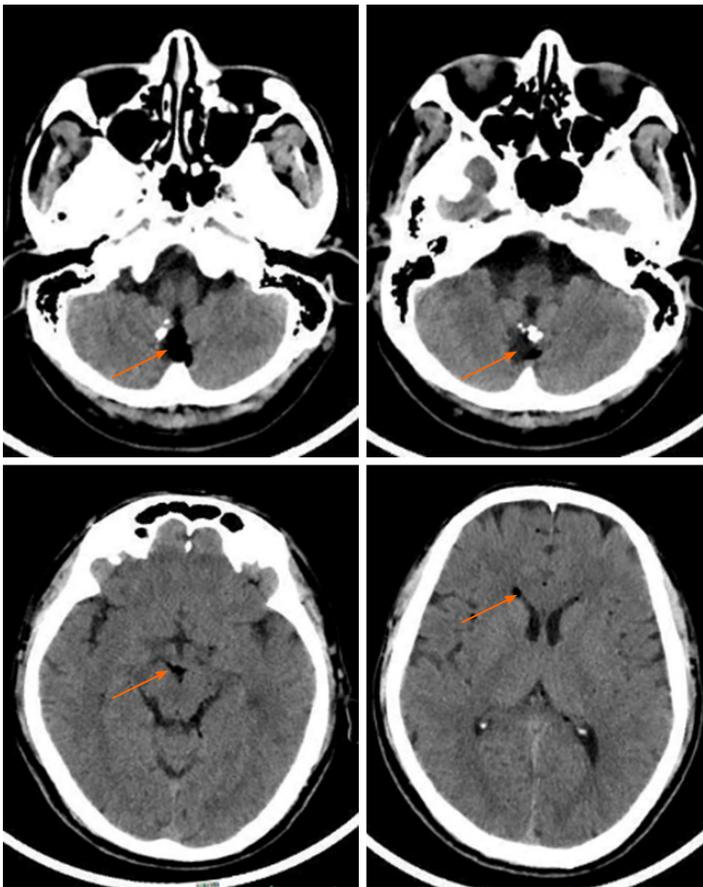


Figure 1 Cranial computed tomography scan showed a mixed-density lesion in the midline of the posterior fossa (arrow), with a fat-density area inside and calcified margin (arrow), as well as lipid droplet drifts in sulci, cisterns, and lateral ventricles (arrows).

enhanced scan did not show any abnormal enhancement in the cystic lesion area or other brain tissues or meninges, and that the fat area presented a slightly hyperintensity and was connected to the subarachnoid space (Figure 2). Interestingly, no abnormal signal was observed in other brain tissue sequences.

FINAL DIAGNOSIS

According to the CT and MRI findings, the patient was finally diagnosed with traumatic rupture of a posterior fossa dermoid cyst with lipid droplet drifts.

TREATMENT

As the patient had no obvious neurological symptoms, he was discharged and sent home for recuperation. As no positive imaging findings or abnormal neurological symptoms were found after 2 wk of conservative observation, a 6-month follow-up visit was arranged for the patient instead of aggressive treatment.

OUTCOME AND FOLLOW-UP

After 1 mo, the patient underwent a neurological examination, which showed no positive findings, no nausea, vomiting, headache, or epilepsy, no abnormal changes in mental state, and normal vital signs and laboratory test results. The patient was proclaimed well in the follow-up visits for up to 6 mo.

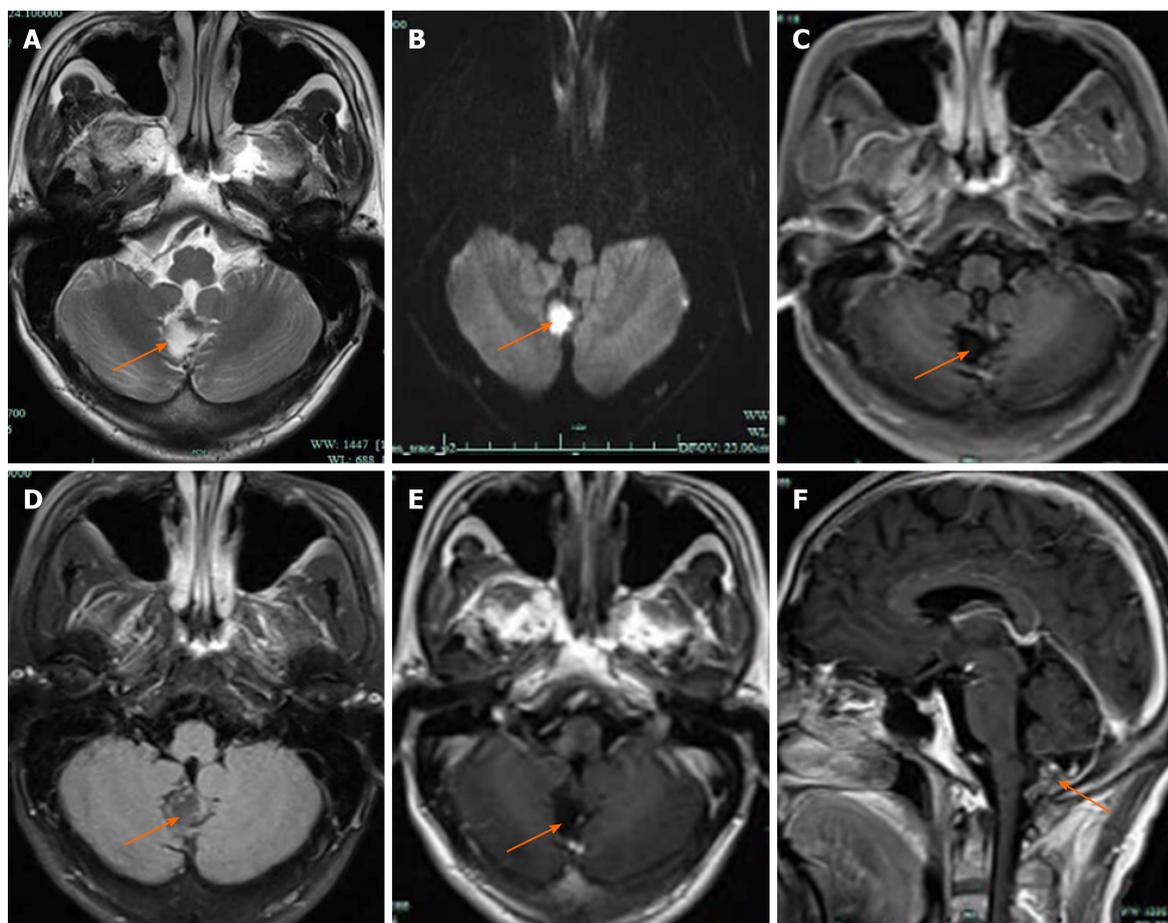


Figure 2 Cranial magnetic resonance imaging at 2 wk after injury. A: The lesion presented hyperintensity on axial T2-weighted imaging (arrow); B: The lesion presented hyperintensity on diffusion weighted imaging (arrow); C: The lesion presented hypointensity on T1-fluid attenuated inversion recovery (FLAIR) (arrow); D: The lesion presented hypointensity on T2-FLAIR (arrow); E: No enhancement in the cystic lesion area was observed on axial T1-FLAIR (arrow); F: On the enhanced sagittal T1-FLAIR, no enhancement was observed in the cystic lesion, while the fat area presented hyperintensity and extended to the subarachnoid space (arrow).

DISCUSSION

Dermoid cysts are epithelial cells remaining in the neural crest ectoderm during 3-5 wk of embryonic development that are wrapped with the closure of neural tubes. Eventually, the cysts are formed that are primarily located in the sellar region, juxtaseellar region, juxta-cavernous sinus, lateral fissures, brainstem, posterior cranial fossa, juxta-fourth ventricle, anterior cranial fossa bottom, and spinal canal[7-9], and larger cysts grow across the cranial fossa[10]. With the advancing age, hormonal changes lead to increased secretion of glands, resulting in the rapid growth of tumors and spontaneous ruptures. When a patient is in his/her adulthood, the hormone level stabilizes. The dermoid cyst is less affected by the hormone and stops growing. In this case, the patient was 59 years old, and had no neurological symptoms before head trauma, suggesting that the lesion was stable. The reason for the dermoid cyst rupture after trauma might be damaged cyst wall and lipid spill caused by the mechanical stress of the internal occipital protuberance on the lesion due to external force to the posterior fossa.

There are two main reasons for the clinical symptoms caused by dermoid cyst rupture. One is the effect of the lesion on the surrounding structure, and the other is aseptic meningitis, vasospasm, and nerve injury caused by the stimulation of the split contents on local meninges, blood vessels, and nerves[10,11]. In this case, no obvious neurological symptoms were observed in the patient before and at the time of injury and during the 6 mo of follow-up visits, which might be related to the location of the lesion below the cerebellar vermis, with no adjacent major nerve or blood vessel. Also, the lesion was small and hence did not exert any obvious compression on the surrounding structure and produced a few split contents after rupture.

In this case, the dermoid cyst extended into the fourth ventricle in the front and connected to the subarachnoid space in the lower part, such that the rupture resulted in extensive drifts of lipid droplets. Although widely distributed, the lipid droplets were small in size, with a maximum length of 5 mm, affecting the local structure slightly and not causing any obvious symptoms. The lipid droplets are difficult to move once they enter the subarachnoid space. However, their extension showed neither absorption of such drifted lipid droplets nor further spread or displacement. The patient did not develop any new neurological symptoms or other adverse clinical consequences[12]. Thus, no special treatment was required for these small lipid droplets according to the study results of the case and literature.

Intracranial dermoid cyst rupture has typical imaging characteristics. In this case, a mixed-density shadow was detected below the cerebellar vermis on CT scan. Furthermore, MRI confirmed the nature and characteristics of the lesion, such as the lipid signal, absence of enhancement area, and limited dispersion, which further affirmed our diagnosis. MRI is superior to CT in determining the correlation between a tumor and the adjacent neurovascular structures, as it excludes the possibility of cerebral infarction, cerebral contusion, and aseptic meningitis, providing a direction for further treatment. Some traumatic or spontaneous dermoid cyst ruptures were treated conservatively with glucocorticoid drugs, which were sufficiently efficacious[13]. In this case, after comprehensively analyzing the imaging and neurological examination results of the patient, the neurologist did not recommend any aggressive treatment, and the patient was declared well throughout the 6-mo follow-up visit.

CONCLUSION

Traumatic intracranial dermoid cyst rupture without neurological symptoms is rare and can be accurately assessed by detailed imaging examinations. For a traumatic intracranial dermoid cyst rupture, if the lesion is small, affects the adjacent structures only slightly, and has few lipid droplet drifts, conservative observation or follow-up visits could be adopted according to the patient's neurological examination results.

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